FOREIGN ANIMAL
DISEASE REPORT

July-August 1978

AFRICAN SWINE FEVER - A REVIEW

African swine fever (ASF) has recently spread to Brazil and the Dominican Republic. The disease was confirmed in Brazil on May 31, 1978, and the Dominican Republic on July 8, 1978.

Technical advisors from the Animal and Plant Health Inspection Service (APHIS) and the Plum Island Animal Disease Center (PIADC) arrived in Brazil on June 4, 1978, at the request of Brazilian animal health officials. Technical advisors and consultants were requested by and sent to the Dominican Republic. The Dominican Republic has announced and is conducting an eradication program consisting of slaughter and disposition of infected and exposed swine. Based on this new and distinctive threat and the often chronic and sometimes inapparent nature of cases, it is essential that each suspicious case be reported and investigated promptly. In order to establish an awareness of this threat and to emphasize the changing nature of ASF, it is being reviewed in this report.

African swine fever is an acute, febrile, highly contagious viral disease of swine. It is considered the most deadly of swine diseases. Most swine contacting this disease die quickly. A chronic or subclinical form, however, is appearing throughout the world which has a milder appearance with lower mortality. African swine fever closely resembles and is often confused with hog cholera (classical swine fever) even though it is caused by an unrelated virus. In Africa, an inapparent infection occurs in the wart hog, bush pig, and other wild swine which serve as carriers and transmit the infection to domestic swine. Surviving domestic swine also serve as carriers and transmit the infection to other domestic swine. Human and other species of animals (including the American Javelina) are not susceptible to ASF. The ASF virus is also transmitted through succeeding generations of Argasid ticks that remain infectious to swine.

History . . . The disease was first recognized in 1909, in Kenya, East Africa. Mortality with ASF frequently approaches 100 percent. From 1909 until 1912, 15 outbreaks were reported. From 1931 to 1961, approximately 60 outbreaks occurred in Kenya. Outbreaks continue to occur in Kenya and in various parts of sub-Saharan Africa. It became a matter of world concern when the disease appeared in Portugal in 1957, and in Spain in 1960. Outbreaks continue to occur in these areas. In 1964, and again in 1967, outbreaks occurred in France. France launched a successful eradication program through quarantine and slaughter. This disease occurred on the island of Madeira in 1966, and in Italy in 1967. In June 1971, ASF appeared

in the Western Hemisphere in Cuba. Slaughter of all swine in Havana Province eliminated the disease. This involved the slaughter of approximately 460,000 swine followed by cleaning and disinfection of all premises. In February of 1974, ASF again appeared on the island of Madeira and the affected pigs were destroyed. In France, outbreaks occurred on the southern frontier in 1974, and were eradicated In late 1977, Spain and Portugal experienced a dramatic increase of cases. In March of 1978, the disease was reported on the island of Sardinia, Italy, and also on the island of Malta.

By June 15, 1978, Brazil reported ASF and by that date 3,654 swine had been slaughtered. This disease is still in Brazil at this time. In early July the Dominican Republic requested assistance from the USDA concerning an outbreak of ASF there.

Signs . . . Hog Cholera, ASF, salmonellosis, and erysipelas are so similar that it is difficult to tell the difference by observing sick animals. Early in an outbreal of any one of these diseases, signs may go unrecognized until several animals have died. Swine usually develop ASF 5 to 9 days after exposure.

Infected swine first undergo an abrupt rise in temperature, although this often goes unnoticed because other signs are usually absent during the early stage of the disease. Occasionally the pigs seem to "burn up" with fever. Swine with acute ASF generally die 4 to 7 days after onset of fever. A day or two before death, the temperature will decrease dramatically. Swine with ASF often retain an appetite while those infected with hog cholera usually do not and die without regaining their appetites. At necropsy ASF generally looks like a severe type of hog cholera.

Transmission . . . New outbreaks may spread fast, especially in cases of the acute form of the disease. Once ASF becomes established a milder form of the disease often develops, as has been the case in Portugal and Spain. ASF is spread by: contaminated garbage, feed, or water; contact between infected and susceptible swine; carrier animals; contaminated premises; clothing, footwear, equipment; improper disposal of infected carcases; and movement of exposed or infected animals from one place to another. Aerosol transmission has also been shown to occur.

Arthropod Vectors . . . Knowledge is incomplete as to the role of arthropods as vectors of the ASF virus. Although primary spread among domestic swine is apparently by contact, three species of arthropods have been incriminated as natural vectors in endemic areas. Ticks probably are of importance in transmitting the ASF virus between wild and domestic swine and can serve as reservoirs of the virus. The hog louse may serve as a vector between domestic swine. Therefore, arthropods appear to play some role in maintaining the disease in wild swine and in serving as a reservoir. Ornithodoros moubata is capable of transmitting ASF virus, but large numbers of ticks are required. This species retains the virus for a relatively short period of time (at least 21 but less than 73 days). Large numbers of ticks collected from the burrows of warthogs suggest that the transmission of ASF virus among warthogs could well depend on the large number of O. moubata infesting swine burrows. In addition, single O. moubata nymphs, experimentally infected with ASF virus, are capable of transmitting the virus for at least 3 months. In 1970, Transovarian transmission was obtained from female O. moubata porcinus. It has been concluded that transovarian infection of the tick is one of the natural maintenance mechanisms for ASF virus. Another investigation showed that Ornithodoros erraticus was capable of transmitting ASF virus between domestic swine for as long as 6 to 12 months.

ASF virus survives in the hog louse (Haematopinus suis) for at least 42 days and possibly during the entire life of the louse, and may be transmitted to other swine. Lice collected from a pig having died of ASF transmitted the virus to susceptible pigs.

There is also the possiblity of mechanical transmission by biting flies. Although this aspect of ASF virus transmission has not been extensively investigated, experience of hog cholera and other viral diseases of livestock suggests the possibility of at least mechanical transmission by tabanids and other biting flies (such as stables flies, black flies, and buffalo gnats, etc.)

Vaccine . . . Despite years of experimentation, extending back to the early years of the century, a satisfactory ASF vaccine has not been developed. Work, however, is continuing along this line at the Plum Island Animal Disease Center (PIADC) of the United States Department of Agriculture where research on foreign diseases is conducted under maximum security. The ASF virus has been propagated in rabbits, embryonated chicken eggs, and various tissue cultures, but a reduction of virulence to a safe level has not been accomplished by passage. Since the ASF virus does not produce neutralizing (protective) antibodies, animals with virus remain shedders and a constant threat to susceptible swine.

Virus Resistance . . . ASF virus is exceptionally resistant. In experiments, blood was still infective after 18 months at room temperature and after 6 years at approximately 40°F. (5°C). Presence of virus in the blood is a consistent feature and, thus, all tissues and organs contain virus. During the acute phase, the blood routinely contains in excess of 1 million lethal doses per milliliter.

ASF virus is inactivated in 20 minutes at 60°C (140°F) but heating at 56°C (132°F) for 30 minutes, the conventional method for inactivating sera prior to testing, does not completely inactivate the virus. The pH stability of the virus at alkaline pH is exceptional. It has been shown to remain viable for 2 hours or longer within a pH range of 1.9 to 13.4 with some strain variation being evident. ASF virus is readily inactivated by lipid solvents and ortho-phenyl-phenol disinfectants.

Laboratory Tests . . . A test which involves the "clumping" of red blood cells around infected white cells in a test tube indicates ASF is present. This test was developed by PIADC scientists working at the East African Veterinary Research Organization Laboratory in Kenya. The practical value of this test was demonstrated during a USDA-supported research project in Spain where an examination of more than 20,000 field specimens clearly established it as a valuable diagnostic tool.

References Available . . . PA 817, "African Swine Fever", may be obtained by requesting a copy from:

APHIS Information Division
U.S. Department of Agriculture
Washington, DC 20250

... A 16mm movie "African Swine Fever - Hog Cholera is available from: the area offices or

Emergency Programs
USDA, APHIS, Veterinary Services
Federal Building
Hyattsville, MD 20782

... A bibliography of citations on ASF may be obtained by requesting a copy from:

Emergency Programs
USDA, APHIS, Veterinary Services
Federal Building
Hyattsville, Maryland 20782

RIFT VALLEY FEVER

Rift Valley Fever (RVF) is an acute, febrile, insect-borne viral disease of sheep, cattle, goats, and humans. The disease is severe, yet rarely fatal in humans. Experimentally, the donkey, mouse, ferret, white rat, and hamster are susceptible. Puppies less than 7 days old are extremely susceptible while adult dogs exhibit few overt signs of the disease except abortions or stillborn litters. Camels also appear to be susceptible with abortions and, in the young, death is often seen The horse, pig, guinea pig, and chicken have been found insusceptible.

Until 1977 RVF had remained limited to the African Continent (Central and South Africa) where it is fairly widespread. In 1950-51, a great epizootic of RVF occurred in South Africa during which it is estimated that 20,000 persons were infected, and 100,000 sheep and cattle died from the disease. Except for several cases in laboratory workers in the United States, Europe, and Japan, the disease previously was not reported other than on the continent of Africa. In the summer and fall of 1977 an explosive, unexpected, and enigmatic outbreak of RVF occurred in Egypt. RVF had never been recognized before in Egypt and its clinical manifestations were totally new to both the veterinarians and physicians in the area. Investigators have reported that the disease in animals, especially abortions in sheep were common during the summer and fall; however, it wasn't until October before human cases were reported in epidemic numbers. Some reports estimated that about 20,000 people were sick and that 70-80 deaths occurred. Other reports indicate these figures were conservative and the number of persons affected was significantly higher.

The virus of RVF is approximately 30-94 nm in size; inactivated at 56°C (133°F) for 40 minutes; stable within a range of pH7-8; and rapidly inactivated at a pH below 6.2. The reservoir for this virus apparently exists in enzootic foci in forested areas with a cycle involving mosquitoes, wild animals, and possibly rodents. Culicine mosquitoes are considered to be the most common natural means of transmission between animals. Humans often become infected secondarily by direct contact with diseased animals especially when handling animal tissues during slaughtering operations, necropsies, and laboratory procedures. During the recent outbreak of RVF in Egypt, the primary method of transmission among animals and humans appeared to be by vectors, primarily the Culex Pipiens. RVF virus is also present in milk at low titer; therefore spread of infection by milk is a possibility. The disease is not believed to be transmitted directly from human to human. Infective mosquitoes probably transmit virus throughout their life. Viremia, essential to vector infection, is present during early clinical illness in humans. The stability of virus aerosols and their extreme infectivity by inhalation indicates the respiratory route to be a significant probability during the handling of diseased carcasses. The incubation period varies from 12-96 hours in sheep and cattle and 4-6 days in humans.

Signs manifested in animals are fever, listlessness, anorexia, unsteady gait, mucopurulent nasal discharge, diarrhea,and abortion. Abortion often is the only clinical finding in adult animals. The mortality in lambs is high, but rarely exceeds 30 percent in adult sheep. In cattle the death rate is lower. In humans, RVF most often assumes an influenza-like character, i.e., myalgia, fever (diphasic curve), headache, weakness, arthralgia, conjunctival congestion, photophobia, icterus, and occasionally vomiting. A leucopenia is found during the acute phase of the illness. Although RVF, as manifested in humans, is normally of high morbidity and low mortality, the complications may be extremely serious. Temporary or permanent blindness often results from macular degeneration and encephalitis has been reported. Death in humans usually results from enlarged livers and spleens.

The salient postmortem feature is widespread areas of focal necrosis and inflammation of the liver with softening and hemorrhages. Widespread subserosal hemmorrhage, enlarged spleen, and gastroenteritis are also seen. Postmortem decomposition is usually rapid.

Control methods are:

1. Protect humans and animals against insect vectors. Establish a large scale vector control program.
2. Establish quarantine areas where livestock are involved.
3. RVF infected animals should not be slaughtered for human consumption.
4. Avoid the handling of infected meats.
5. Utilize vaccine in the animal population.

BIRD IMPORTATIONS

Since 1972, the U.S. Department of Agriculture (USDA) has regulated the importation of exotic birds into the United States when it was determined that such importations were introducing exotic Newcastle disease. Initially, August 1972 to October 1973, the control of such imports consisted of a total prohibition on commercial importations. Following the prohibition, the regulations were amended to allow importations under specific conditions. These conditions were designed to prevent the introduction of communicable disease of poultry as the USDA's authority is limited by law to this area of disease control. The responsibility for preventing the introduction of psittacosis rests with the U.S. Department of Health, Education, and Welfare. Cooperation between this Department, as well as the U.S. Customs Service and the U.S. Department of the Interior, exists in order that the various agencies can meet their legal responsibilities.

The first shipment of birds to be released under this program occurred in January 1974. Since that time, over 700,000 birds have entered the United States. In FY 1977, over 300,000 birds were released for entry, while 35,000 were refused entry. All the birds refused entry were due to a diagnosis of velogenic viscerotropic Newcastle disease. No lots were determined to be positive from November 1977 to May 1978. This is the longest period of time to elapse without a group of birds being found to be harboring the exotic Newcastle virus. Other viruses have been isolated from imported birds. One of the more significant viruses is herpes virus, which is associated with Pacheo's disease. Viruses,

which are not characterized as a Newcastle virus, are inoculated into four test turkeys and four test chickens. If no clinical signs of disease are observed, then the virus is considered to be nonpathogenic for poultry and the shipment of birds, if otherwise eligible for entry, are released. To date, this program has proven to be successful as no known cases of Newcastle disease can be attributed to legally imported birds.

EGG DROP SYNDROME

During the autumn of 1976, a new syndrome, now called the egg drop 1976 (EDS76) syndrome, was recognized in Northern Ireland. It is now clear according to a paper presented by Dr. J. D. McFerran, Department of Agriculture, Belfast, that outbreaks of the same syndrome occurred in England early in 1976, and a similar condition was present in the Netherlands for some years.

The first signs of depressed production of usable eggs were seen in birds 26-35 weeks of age. This should be contrasted with most infectious diseases, such as infectious bronchitis and Newcastle disease, where susceptible flocks of any age show signs.

The major effect was on the numbers and quality of the eggs. There were falls in production of 10-40 precent immediately after reaching peak production.

During an investigation into the etiology of EDS76, six viruses belonging to one adenovirus serotype were isolated. One isolate, 127, was selected for further study.

The epidemiology of this condition is difficult to understand. The EDS76 Syndrome and antibody to 127 virus would indicate that this virus has unconventional spreading abilities. Thus it was distributed throughout Northern Ireland within weeks of its first appearance. However, birds belonging to different organizations, even when in relatively close contact, have not been affected, indicating either a resistance to infection or lack of lateral spread.

Provisions have been made through the Import Staff of Veterinary Services to allow killed antigen to be used by several requesting laboratories. Serologic surveys should indicate whether antibodies to adenovirus 127 are present in poultry populations in this country.

BOVINE HERPES MAMMILLITIS

Animal health officials are being urged to be on the alert for symptoms of bovine herpes mammillitis (BHM).

BHM antibodies have been demonstrated in the States of Iowa, Louisiana, Minnesota, New York, South Carolina, Tennessee, and Virginia. Suspicious cases of BHM are being reported more frequently throughout the United States. It is of extreme importance to continue reporting these symptoms since it is similar in many respects to lumpy skin disease, a disease exotic to the United States. A laboratory differential diagnosis should be requested in all suspicious cases.